

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re application of:
R. Wright. *et al.*
Appl. No. 10/573,354
Filed: 10 May 2007
For: **Methods of Modulating
Inflammatory Reactions by Modulating
Xanthine Oxidoreductase Activity**

Art Unit: 1613
Examiner: S.M. Basquill
Atty. Docket: 000115-5002
Confirmation No.: 2805
Customer No.: 09629

RESPONSE TO FINAL OFFICE ACTION

This paper responds to the Final Office Action of 23 November 2010.

Anticipation

The Office Action of page 2 rejected claims 1-3, 5, 7-12, 14, 16-18, and 20 for allegedly being anticipated under 35 USC § 102(b) by Chabot.

The Office Action on page 3 alleges that the step of selecting for an acute lung injury would necessarily require selecting a patient suffering from inflammation caused by increased XOR activity. Applicants respectfully disagree. Chabot provides no teaching regarding XOR activity, and the Examiner has cited to no reference that supports the allegations that acute lung injury necessarily requires selecting a patient with increased XOR activity. It is only through Applicants own work that the step of selecting for increased XOR activity becomes apparent.

As acknowledged by the Examiner on page 3, Chabot teaches ischemia-reperfusion injuries. Chabot is silent with regard to selecting for increased XOR activity in leukocytes and leukocyte precursors. Chabot is silent with regard to any XOR activity. Increased XOR activity in leukocytes and leukocyte precursors is not a necessary effect and it is not always inherently present in ischemia-reperfusion injury: the two are not synonymous. The burden is on the Office to prove that increased XOR activity in leukocytes and leukocyte precursors is an inherent and necessary consequence of ischemia-reperfusion injury. The Office has failed to carry its burden.

Similarly, inflammation from increased XOR activity in leukocytes and leukocyte precursors is not necessarily only associated with reperfusion injury. Accordingly, the step of selecting for XOR activity in leukocytes and leukocyte precursors is different to the ischemia-reperfusion injuries of Chabot. In fact, the step of selecting for increased XOR activity is a marked improvement over Chabot as it precisely identifies those that would actually benefit from allopurinol treatment. As identified in the present application, allopurinol's effects are much more marked when XOR activity is increased in leukocyte and leukocyte precursors.

Chabot fails to disclose all acute lung injuries. Increased XOR activity in leukocytes and leukocyte precursors is not a necessary effect and it is not always inherently present. The Office must prove that increased XOR activity in leukocytes and leukocyte precursors is an inherent and necessary consequence of all lung injuries. The claimed invention provides a selection step that improves treatment with XOR inhibitors by ensuring that XOR activity is imbalanced in leukocytes or leukocyte precursors. This selection step is not taught or necessarily present in the methods of Chabot. Accordingly, Chabot does not disclose every feature of the claimed invention, and therefore cannot anticipate the claimed invention.

With regard to MPEP 2111.02, Applicants submit that the Office Action misinterprets this rule and inappropriately misapplied this section to the present claims. MPEP 2111.02 refers to the preamble of a claim for an intended use, however, Applicants claimed invention recites the active step of selecting for increased XOR activity, which is an element in the recited method. As discussed above, the step of selecting for XOR activity is not taught by Chabot. Accordingly, the claimed invention is not anticipated.

Should the Examiner believe that further discussion of any remaining issues would advance the prosecution, he or she is invited to contact the undersigned at the telephone number listed below.

Response to Non-Final Office Action
U.S. Serial No.: 10/573,354
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Atty. Dkt. No. 000115-5002

Respectfully submitted,

Date March 23, 2010

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